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PHYSIOLOGIC BASIS FOR CO_2 LIMITS WITHIN SEMICLOSED- AND CLOSED-CIRCUIT UNDERWATER BREATHING APPARATUS

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19. ABSTRACT:

Semiclosed- and closed-circuit underwater breathing apparatus (UBA) incorporate a canister filled with carbon dioxide (CO_2) absorbent material. As the absorbent becomes expended, the CO_2 level within the breathing loop will rise. Currently, CO_2 absorbent canisters are considered expended when the effluent CO_2 is 0.5% Surface Equivalent Value (SEV). In the past, this value often represented the threshold between the linear and the exponential rise in effluent CO_2. The 0.5% SEV limit was used to reduce the diver's risk for experiencing excessive levels of CO_2. However, with advances in absorbent canister design, this value may be overly conservative. Because the overall respiratory load of a UBA can compound the effect of the inspired CO_2, the improved breathing performance of some current UBAs also warrants reevaluating the current limit. This literature review specifically looked at low levels of CO_2 affecting work and mental performance, nitrogen narcosis, decompression sickness and central nervous system (CNS) oxygen toxicity to determine if the breakthrough limit could be changed.

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19. Abstract continued:

RESULTS. A reduction in the maximal work capacity was observed when the inspired PCO_2 was 2% and greater. No deterioration in psychomotor and mental performance was detected for inhalation of up to 5% CO_2 during exercise. However, alveolar CO_2 tensions in excess of 40 mmHg (test conditions used an inspired CO_2 of 2%) may potentiate the effect of nitrogen narcosis. This factor is an important consideration for the full operational usage of the MK 16 with the 0.7 ATA of oxygen in nitrogen. Breathing CO_2 greater than 1% for over 1 hour has been associated with an increased the risk for decompression sickness. Two-percent CO_2 may increase the onset of an oxygen convulsion when combined with elevated percentages of oxygen because of the increase in brain capillary blood flow.

CONCLUSION: The literature suggests that a short duration exposure, 15 minutes, to a CO_2 level of 2% SEV will not result cause a catastrophic effect on a diver. The literature suggests that a CO_2 level of 2% produces a minimal effect on a diver's physical and mental work performance. At most, there may be a marginal increase in the risk of decompression sickness. The risk of CNS oxygen toxicity does not appear to be increased for U.S. Navy oxygen diving operations. When designing CO_2 scrubber canister limits based upon the above described physiological response, the UBA's respiratory load and canister carbon dioxide absorption characteristics, oxygen or breathing gas supply duration also must be considered.

ACKNOWLEDGEMENT

I thank Dr. Chris Lambertsen M.D., "Father of U.S. Combat Swimming," for his review of this paper's technical accuracy and the appropriateness of the derived conclusions based upon the presented literature and his experience.

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PHYSIOLOGIC BASIS FOR CO_2 LIMITS WITHIN SEMICLOSED- AND CLOSED-CIRCUIT UNDERWATER BREATHING APPARATUS

INTRODUCTION

Semiclosed- and closed-circuit underwater breathing apparatuses (UBAs) incorporate a canister filled with carbon dioxide (CO_2) absorbent material. This design efficiently uses the gas supply because a portion of the diver's exhaled breath is re-circulated through the system. Additional gas is added to the UBA to replace the oxygen consumed by the diver. As the absorbent becomes expended, the CO_2 level within the breathing loop will rise. Because CO_2 does have a physiologic effect that may affect a diver's performance, the question becomes 'how much CO_2 is too much.'

Historically, canister limits were defined as the time it takes in minutes or the liters of CO_2 absorbed until the canister effluent gas CO_2 reaches 0.5% Surface Equivalent Value (SEV)[1,2]. During the current evaluations of the MK 25, a closed-circuit UBA, the SPECWARCOM Biomedical Research and Development Medical Officer raised the issue that this value for the partial pressure of CO_2 (P_{CO_2}) limit may be unduly conservative. The purpose of this paper is to review the literature and recommend a canister limit based upon the known data of the physiologic effects of low levels (<4%) of CO_2.

METHODS

The primary concerns of low levels of CO_2 are their possible effects on a diver's mental and physical performance. However, this discussion must also address a UBA's breathing resistance, since hypoventilation will also increase the diver's blood CO_2 level and potentiate any CO_2 problem. Furthermore, high levels of CO_2 in the blood will change the diver's blood chemistry affecting work performance. Carbon dioxide also affects cerebral blood flow that may influence the diver's threshold to Central Nervous System (CNS) oxygen toxicity. In addition, the role of CO_2 on decompression sickness must also be addressed. This literature review primarily concentrated on the articles frequently quoted when discussing the effects of CO_2. It must be realized that the studies involving CO_2 levels below 3% are sparse. Typically, studies used at least 5% CO_2 to ensure a physiologic effect could be observed.

DISCUSSION

INTERRELATIONSHIP OF RESISTIVE LOADS AND CO_2

To understand the physiologic effects of inspired CO_2, its relationship to breathing with an underwater breathing apparatus must be understood. The following mathematical formula shows the relationship between the factors affecting the alveolar

CO_2 levels. In normal individuals, the alveolar PCO_2 (P_ACO_2) is equal to the arterial PCO_2 (P_aCO_2). It's the blood CO_2 that causes the physiologic effects.

$$P_ACO_2 = P_ICO_2 + 863\ \dot{V}_{CO_2} / \dot{V}_E\,(1 - V_D/V_T) \qquad \text{Equation (1)}$$

Where:

P_ACO_2 - alveolar PCO_2 (mmHg)
P_ICO_2 - inspired PCO_2 (mmHg)
863 - factor for correcting $\dot{V}$ from STPD to BTPS
$\dot{V}_{CO_2}$ - minute volume of CO_2 (liters per minute; STPD)
$\dot{V}_E$ - minute ventilation (liters per minute)
V_D - dead space volume (liters)
V_T - tidal volume (liters)

Any carbon dioxide inspired will directly affect the blood CO_2 levels. In addition, any impediment to ventilation will cause the P_aCO_2 to rise. For example:

$\downarrow \dot{V}_E$ when: $\uparrow$ gas density
$\uparrow$ rig resistance
$\uparrow$ inspired oxygen during exercise

Ventilation increases with increasing exercise. When the inspired fraction of CO_2 rises, there is a concomitant rise in ventilation[3]. Figure 1 illustrates the rise in minute ventilation with increasing work rates. When CO_2 is added to the inspired gas, the ventilation rate further elevates above the level expected from exercise alone.

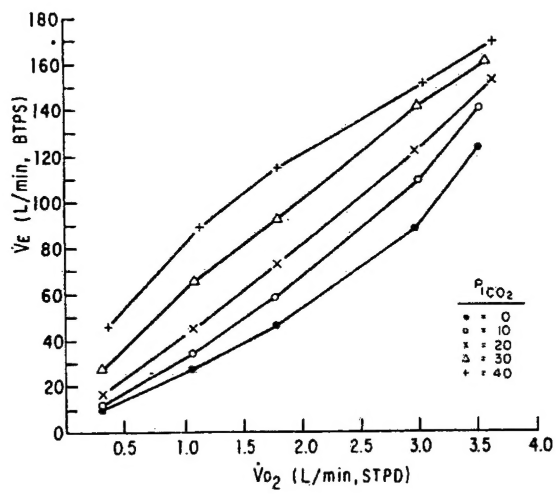

Figure 1. Relationship of ventilation to O_2 uptake during exercise at different levels of inspired CO_2 tension. The average values of oxygen uptake ($\dot{V}_{O_2}$) at rest and at 4 different work loads were not significantly altered by changes in P_ICO_2.

Reprinted from: Clark J. M., Sinclair, R. D. and J. B. Lenox. Chemical and nonchemical components of ventilation during hypercapnic exercise in man. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 48(6): 1065-1076,1980.

Clark et al., further demonstrated that the level of inspired CO_2 affects the arterial CO_2[3]. For exercise performed on the surface breathing air, a person actually hyperventilates during exercise performed at 50% of maximal oxygen uptake ($\dot{V}_{O_2}max$) and greater. Figure 2 illustrates that when the P_ICO_2 is increased above 20 mmHg, the P_aCO_2 dramatically rises for the level of exercise.

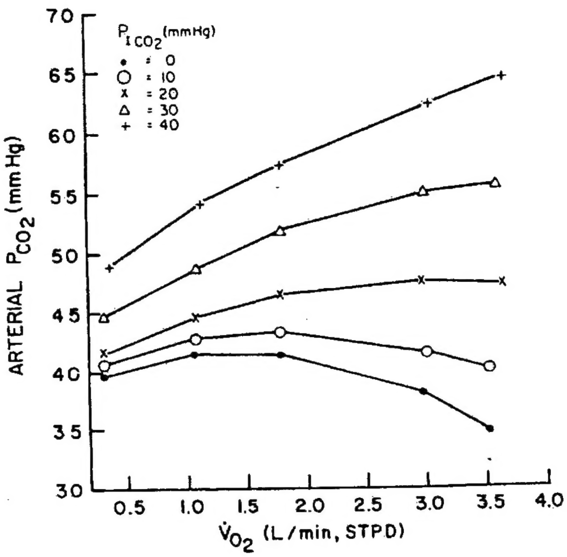

Figure 2. Arterial P_{CO_2} during exposure to combined exercise and hypercapnia.

Reprinted from: Clark J. M., Sinclair, R. D. and J. B. Lenox. Chemical and nonchemical components of ventilation during hypercapnic exercise in man. J. Appl. Physiol.: Respirat. Environ. Exercise Physiol. 48(6): 1065-1076,1980.

Experiments performed by Poon illustrate the ventilatory responses to hypercapnea and exercise when breathing through an inspiratory resistive load (~12 $cmH_2O/l/s$)[4]. The investigator kept the end-tidal P_{CO_2} ($P_{ET}CO_2$) constant, while the subject inspired a constant fraction of ~5% CO_2.

Figure 3 shows that an inspiratory resistive load further reduces minute ventilation when the $P_{ET}CO_2$ exceeds 40 mmHg. Therefore, breathing patterns are a result of a balance of chemical drive and the propensity to reduce respiratory effort.

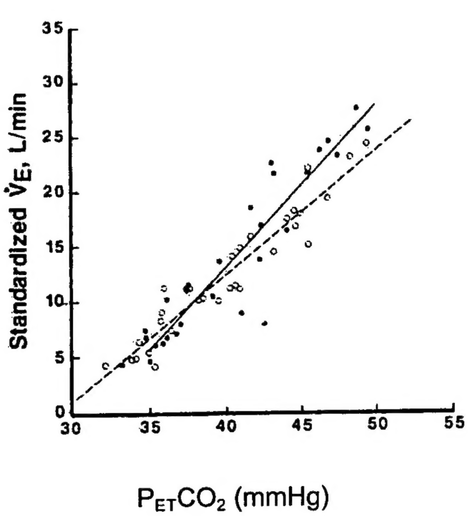

Figure 3. Responses in minute ventilation to $P_{ET}CO_2$ under no load (filled circles, solid line) and inspiratory resistive load ~12 $cmH_2O/l/sec$ (open circles and dashed lines).

Reprinted from: Poon, C. S. Effects of inspiratory resistive load on respiratory control in hypercapnia and exercise. J. Appl. Physiol. 66(5): 2391-2399,1989.

Application of an external resistance will also affect a person's $P_{ET}CO_2$. Demedts and Anthonisen demonstrated that as breathing resistance approaches 15 cmH$_2$O/l/sec, there's a distinct increase in $P_{ET}CO_2$, which increases with exercise load; whereas, at resistances less than 5 cmH$_2$O/l/sec there's no change in the $P_{ET}CO_2$ for the increasing work load[5]. Figure 4 illustrates the effect of resistance on $P_{ET}CO_2$.

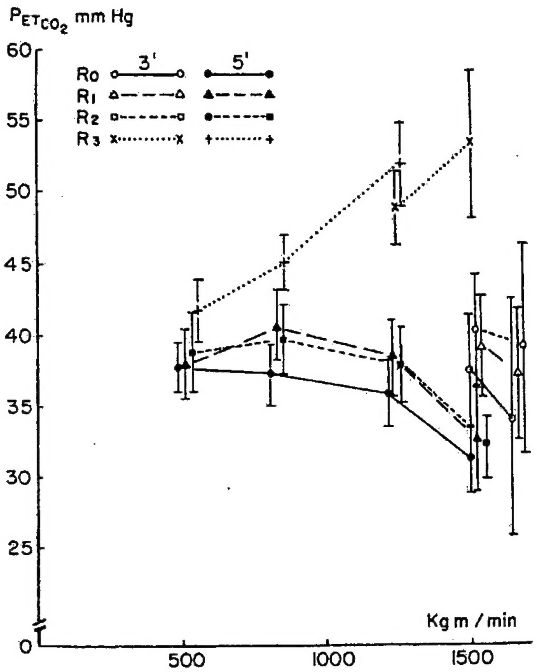

Figure 4. Effect of resistance on $P_{ET}CO_2$. *Ordinate:* end-tidal CO_2 tension (mmHg). *Abscissa*: work load in kgm/min. Mean values ±1 SE are shown for all subjects during the 5th min of exercise and when this was not available, in the 3rd min. The levels of resistance were:

R_0 - none
R_1 ~ 0.5 cmH$_2$O/l/sec
R_2 ~ 1.0 cmH$_2$O/l/sec
R_3 ~ 15 cmH$_2$O/l/sec

Reprinted from: Demedts, M. and Anthonisen, N. R. Effects of Increased External Airway Resistance During Steady-state Exercise. J. Appl. Physiol. 35(3): 361-366, 1973.

In general, when a person doesn't breathe adequately, the P_aCO_2 will rise. This is the case in diving. Divers tend to have a markedly decreased breathing frequency and increased tidal volumes, which is not related to fitness, when compared to non-divers[6]. Also perhaps because of a diver's experience with the desire to conserve breathing gas, the diver's breathing pattern results in a relative hypoventilation, regardless of UBA type. In addition, during exercise, there is a pronounced hypoventilation and hypercapnia in divers[7]. Hence, with the addition of an UBA's breathing resistance, the diver typically will hypoventilate for the work being performed resulting in an increased $P_{ET}CO_2$.

CO_2 EFFECTS ON WORK PERFORMANCE

Overall work performance may decrease with increased inspired CO_2. Work performed in the 1970's for the National Aeronautics and Space Administration established that maximal work capacity is reduced when the P_ICO_2 is 15mmHg (2%)[8,9]. The authors attribute the deterioration in work performance to the interference to eliminating the excess CO_2 from exercising muscles and the resulting acidemia. No decrements were reported for P_ICO_2 less than 2%.

Craig et al., conducted studies where exhausting exercise was performed while inhaling against a resistance with a gas mix containing 3 to 4% CO_2[10]. Figure 5 illustrates that when CO_2 is added to the inspiratory gas with a resistance of 1.5 cm H_2O/l/sec (R1), ventilation increases and the person was able to walk for 10 minutes before becoming exhausted. However, when breathing against a resistance of 15.5 cm H_2O/l/sec (R4) combined with 4.3% P_ICO_2, the person did not produce the appropriate ventilatory response and the time to exhaustion was much shorter. Hence, if the breathing resistance is kept low, there's an adequate response to the inspired CO_2 and work performance is not impacted.

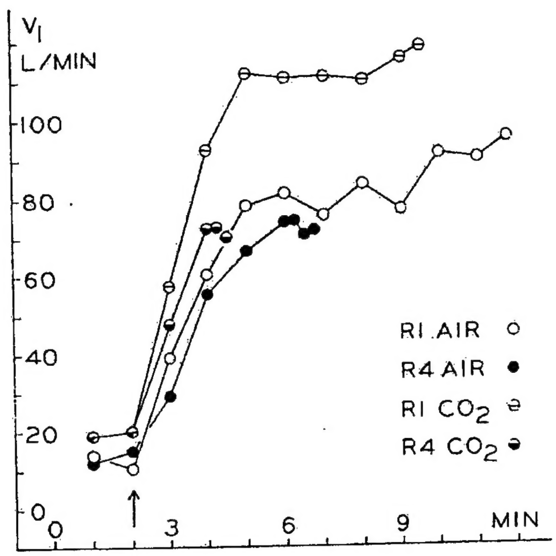

Figure 5. Respiratory minute volume during walks under varied conditions of inspiratory resistance and inhalation of carbon dioxide. Arrow marks beginning of walking. Concentrations of carbon dioxide in inspired air were 3.9% at R1 and 4.3% at R4.

Reprinted from: Craig, F. N. Blevins, W. V. and E. G. Cummings. Exhausting Work Limited by External Resistance and Inhalation of Carbon Dioxide. J. Appl. Physiol. 29(6): 847-851, 1970.

CO_2 EFFECTS ON COGNITIVE PERFORMANCE

The increase in a person's P_aCO_2 is referred to as CO_2 retention. Therefore, the effect of an increased P_ICO_2 must be evaluated in light of an increased P_aCO_2. Henning et al., evaluated the behavioral effects of an elevated P_ICO_2 to clarify the risks due to CO_2 retention[11]. For this study, the subjects breathed 6% CO_2 in 21% and 94% oxygen and performed various psychometric tests. The authors concluded that divers may be at risk for disequilibria, impaired decision making and disturbances in motor control immediately following a period of CO_2 retention. Furthermore, if a diver hypoventilates and if a high breathing resistance is suddenly added, the result can be sudden unconsciousness[12]. Hence, to minimize the potential of hypoventilation and its potentially catastrophic consequences, the breathing resistance within semiclosed- and closed-circuit UBAs should be as low as technically possible.

The breathing resistance of a closed-circuit UBA can increase as the CO_2 absorbent material is depleted. Divers reported that during underwater exercise at 60 feet they had to use the diluent by-pass of the MK 15 to increase gas flow to reduce breathing

effort when the canister effluent CO_2 exceeded 3.8 mmHg (0.5% SEV)[13]. It should be noted that the divers in that study did not report any other symptoms even though the canister effluent CO_2 reached 1.5% SEV. However, theoretically, divers who are breathing at a low ventilation rate because of their breathing apparatus, combined with an increase in inspired CO_2 may run the risk of impaired performance if the resistive load progressively increases. Unfortunately, no controlled studies were performed at NEDU that determined the actual increase in breathing resistance with an increasing canister effluent CO_2 and its affect on ventilation. However, all the studies reviewed only found symptoms when the diver was exposed to very high resistances or a P_ICO_2 of 5% or greater.

A diver's cognitive performance is critical in an underwater environment. Errors in judgement can be catastrophic. Sheehy et al., evaluated the effect of 4% and 5% CO_2 in 21% and 50% oxygen on cognitive performance during exercise and recovery on the surface[14]. They reported no deterioration in psychomotor and mental performance for the inhalation of up to 5% CO_2. The investigators noted that the short-term memory test they used detected effects due to strenuous exercise, whereas low levels of CO_2 did not cause a decrement in memory performance. Recalling Equation (1), if the P_ACO_2 is kept below 5% by appropriate ventilation for the level of P_ICO_2, the diver's cognitive performance would not be limited.

However, the MK 16 with a PO_2 set point of 0.7 ATA in nitrogen does have a significant level of inert gas in its breathing loop at its deep operating depth. In fact, the MK 16 limits its operational depth to 150 fsw because of the significant nitrogen narcosis that a diver experiences, especially under working conditions. Hesser et al., described a relationship between CO_2 and nitrogen narcosis[15]. This study used CO_2 concentrations of 0, 2, 4 and 6% and was performed at the surface and at 6 ATA. The results suggested that when the inspired gas tension of nitrogen (P_IN_2) and the inspired CO_2 (P_ICO_2) rose simultaneously, their effect on performance was greater than the arithmetic sum of the changes induced by either gas alone. Allowing the P_ICO_2 to increase to 2% may potentiate the risk of nitrogen narcosis.

CO_2 EFFECTS ON CENTRAL NERVOUS SYSTEM O_2 TOXICITY

An increase in the P_ICO_2 may reduce the time for the development of Central Nervous System (CNS) oxygen toxicity symptoms. The mechanism for this belief is the fact that CO_2 will cause a cerebral vasodilatation[16] resulting in an increase in brain oxygenation. Figure 6 illustrates the exponential rise in cerebral blood flow when the arterial PCO_2 rises.

As discussed earlier, P_aCO_2 will increase with exercise when breathing against a resistive load. This pattern also is seen when exercising while breathing 100% oxygen at 2 ATA, though the absolute rise for each individual may be different[17]. Figure 7 shows 6 subjects' P_aCO_2 at rest at 2 ATA while breathing 10.5% oxygen in nitrogen, and then during an incremental exercise while breathing 100% oxygen. Prior to starting the exercise, the P_aCO_2 decreased during the transition from normoxia to oxygen breathing

with an associated increase in ventilation. Though a rise in P_aCO_2 increased for all the subjects, the change in the P_aCO_2 at the highest workload ranged from 4.4 to 14.2 mmHg in the individual subjects.

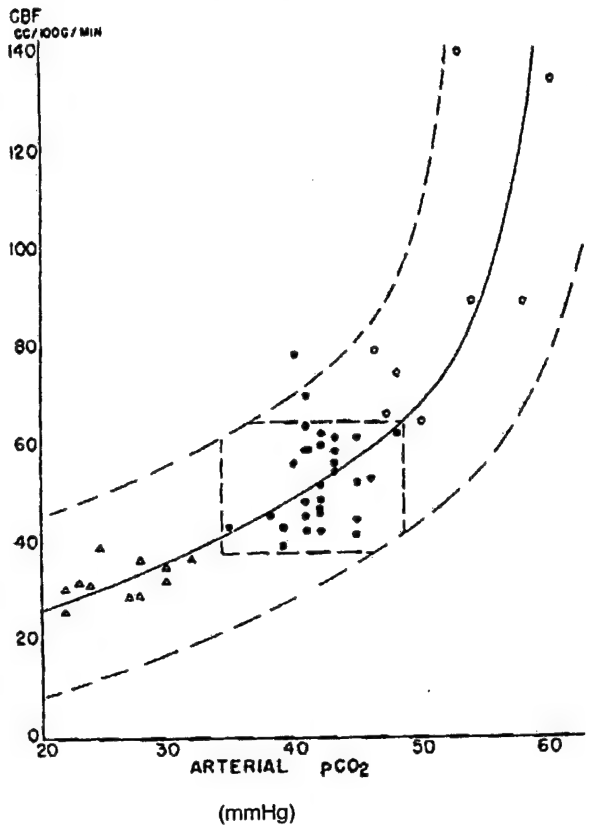

Figure 6. The relationship between cerebral blood flow and arterial CO_2 tension. The arterial PCO_2 varied from the normal (dots) by hyperventilation (triangles) or by inhalation of 5-7% CO_2 (open circles). The dashed lines bound 98% of the observations while the central polygon encloses 94% of the normal values.

Reprinted from: Kety, S. S. and C. F. Schmidt. The Effects of Altered Arterial Tensions of Carbon Dioxide and Oxygen on Cerebral Blood Flow and Cerebral Oxygen Consumption of Normal Young Men. Journal of Clinical Investigations, 1948, 27, 484-492.

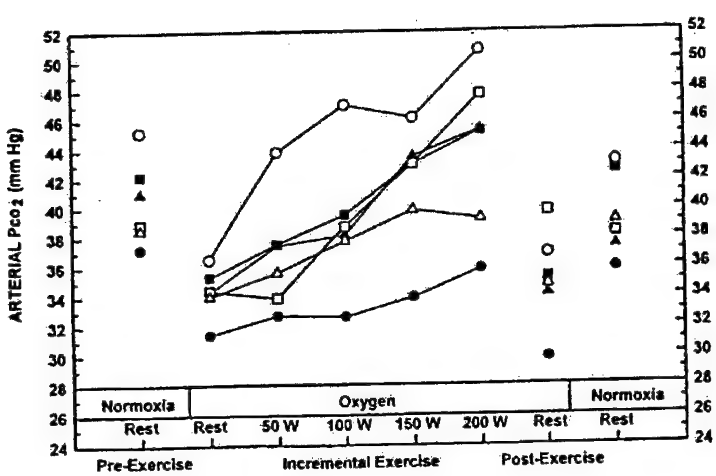

Figure 7. Individual arterial PCO_2 responses to incremental exercise while breathing O_2 at 2 ATA. Data are shown for 6 subjects who completed all 4 workloads.

Reprinted from: Clark J. M., R. Gelfand, C. J. Lambertsen, W. C. Stevens, G. Beck and D. G. Fischer. Human tolerance and physiological responses to exercise while breathing oxygen at 2.0 ATA. Aviat. Space Environ. Med. 1995; 66: 336-345.

The concern is that with an increased P_aCO_2 there is a concomitant increase in cerebral blood flow. With the increase in blood flow, more oxygen can be delivered to the brain, which may result in higher tissue oxygen and a higher direct toxic effect on the neuron, thereby accelerating the onset of an oxygen convulsion. A series of experiments was performed at the Institute for Environmental Medicine at the University of Pennsylvania to evaluate the effect of increased oxygen pressures and carbon dioxide on cerebral blood flow[18]. These studies simulated a variety of conditions: 1) PO_2 of 1, 3 and 3.5 ATA while at rest; 2) PO_2 of 2 ATA while exercising; 3) PO_2 of 3.5 ATA with 2% PCO_2 while at rest. Lambertson reported that only under the last condition, did the brain oxygenation drastically increase from 100 mmHg to 1000 mmHg. Figure 8 illustrates the effect of increased oxygen levels at rest and at exercise, as well as the added effect that high inspired CO_2 partial pressures have on cerebral blood flow.

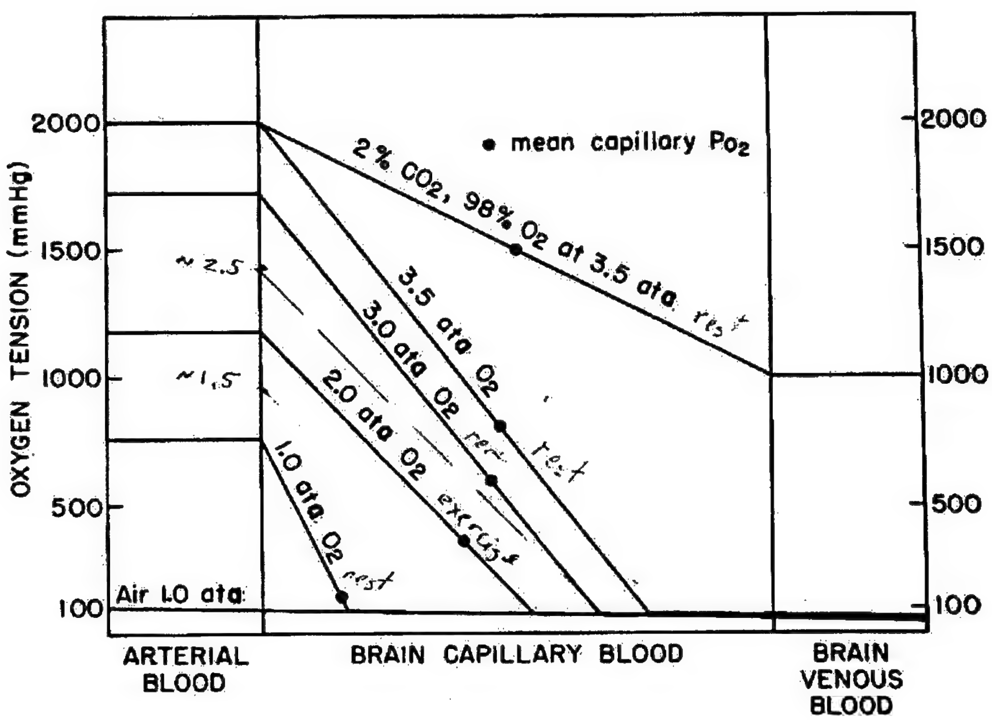

Figure 8. Effect of increased inspired PO_2 on the oxygen tensions of arterial, mean brain capillary, and internal jugular venous blood (average values in normal men). The graph illustrates for each of several levels of inspired PO_2 the manner in which oxygen tension across the mean brain capillary is increased by progressive increases in inspired PO_2. The patterns of change in brain capillary PO_2 are calculated from experimentally determined levels of oxygen pressure in arterial and brain venous blood on the assumption of uniform O_2 loss. When arterial hypercapnia was introduced by administering carbon dioxide with oxygen at 3.5 ATA, brain oxygenation was drastically increased.

Reprinted from: Lambertsen, C. J. Effects of Hyperoxia on Organs and Their Tissues. In: Extrapulmonary Manifestations of Respiratory Disease, ed. Eugene Debs Robin. Vol. 8 of Lung Biology in Health and Disease, ed. Claude Lenfant. New York: Marcel Dekker, 1978.

Because the parameters of the University of Pennsylvania's studies are outside the allowed U.S. Navy limits for oxygen diving, interpolations were made. Assuming that the relationships between the vascular responses are similar for the various conditions as exemplified in Lambertson's report, a diver breathing a PO_2 of 2.5 ATA (100% oxygen at 50 fsw), and inspiring 2% CO_2, the oxygen tension in the brain's venous blood is approximately 400 mmHg. This is a substantial increase in cerebral blood flow and should increase the rate of development of CNS oxygen poisoning.

However, laboratory experiments do not necessarily reflect the conditions experienced operationally. The University of Pennsylvania's experiments bracketed the U.S. Navy Single-Depth Oxygen Exposure Limits, which restricts the exposure of 2.5 ATA PO_2 to 10 minutes. Typically, MK 25 operations are conducted in 20 feet of seawater and shallower. Under these conditions, the low brain venous blood oxygen tension, which reflects the low capillary blood oxygen tension, has a reduced risk of CNS oxygen toxicity. To summarize, if the P_ICO_2 is allowed to momentarily rise to 2%, surface equivalent value, a 15 minute exposure would not increase the risk of CNS oxygen toxicity when diving the U.S. Navy Single-Depth Oxygen Exposure Limits.

CO_2 EFFECTS ON DECOMPRESSION SICKNESS

In 1908 Boycott, Damant and Haldane proposed that an elevated CO_2 would increase circulation to the muscles and increase the elimination of inert gas. This concept favors exercise during decompression. On the other hand, other reports suggest that the opposite is true[19]. Whether to allow exercise during decompression is still a rather controversial area without a definitive conclusion. Therefore, for the purpose of this discussion only the effect of an elevated inspired CO_2 will be considered. This condition closely reflects the operational experience.

One article reported an elevated rate of decompression sickness (DCS) in deep (3.2 ATA (72.6 FSW)) caisson operations in Japan[20]. Due to ventilation problems, CO_2 within the man-lock rose between 1.8 and 2.3%. In this case the incidence of DCS was 3.05%. When the lock was ventilated to reduce the CO_2 between 0.3 and 0.8%, the incidence of DCS was 0.96%. Unfortunately, the authors did not report which particular profiles resulted in DCS. Specifically they did not report the actual CO_2 exposure time for the decompression profile. Therefore, based on this article it's unknown if there's a dose-response relationship of CO_2 exposure to an increased incidence of DCS. The only conclusion is that an elevated CO_2 may result in an increased incidence of DCS.

CONCLUSION

The literature suggests that a short duration exposure, 15 minutes, to a CO_2 level of 2% SEV may not result immediately in a catastrophic effect on a diver. Though there is a distinct relationship of CO_2 to CNS oxygen toxicity with a sustained elevated inspired CO_2, a momentary exposure would not greatly increase the risk of an oxygen convulsion.

Though the MK 16 diver using 0.7 PO_2 in nitrogen may experience narcosis normally during a deep excursion, the operational use of this UBA suggests that when a canister effluent reaches 2%, it would be during a shallow portion of the dive where the PN_2 is relatively low. Hence, it is unlikely that a MK 16 diver would experience a decrement in cognitive performance. There also is little evidence to substantiate the concern that a CO_2 level of 2% SEV greatly increases the risk of decompression sickness.

Our literature review indicates that a diver can tolerate a CO_2 level as high as 2.0% SEV for 15 minutes with minimal risk. However, the current 0.5% SEV limit for CO_2 inhalation was established to allow a margin of error in minimizing the potential that a diver would breathe potentially catastrophic levels (greater than 4% SEV) of CO_2. This was essential since unmanned testing does not simulate all the variability that exists during diving operations. Specifically, unmanned simulations define a specific CO_2 injection rate for a presumed oxygen consumption rate at a particular respiratory minute volume. Furthermore, unmanned testing did not define the canister performance characteristics beyond 1% SEV. Because of this limited testing, the rate of rise in the CO_2 levels can not be ascertained so a safety margin had to be postulated.

It is important to note that canister duration limits should not be based only on the average CO_2 breakthrough curve, which does vary with different UBA, but also on the oxygen or breathing gas supply duration as well as the physiological variations between divers and diving missions. It is possible that in the future once the UBA characteristics and diver variability can be adequately defined, or when a reliable underwater CO_2 monitor becomes available, we will recommend increasing the CO_2 level beyond the current 0.5% SEV.

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